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Research Article

Gastric Ulcer and Canned Materials and Smoking: Case Control Study

Rana Alhalaf, Abdelrahman Alitr*

Department of Internal Mdicine, Mansoura University, Department of Internal Medicine, Mansoura City, Egypt

Abstract

This study aimed to explore the potential role of smoking and canned material consumption in the development of gastric ulcer disease. Researchers compared 104 gastric ulcer patients with a control group matched for age, sex, and social status. The analysis examined exposure to these factors during three periods: over a lifetime, within five years, and in the year preceding the onset of symptoms. Results revealed a significant link between smoking, regular canned material consumption, and a higher risk of gastric ulcers across all time frames. Since these exposures occurred prior to symptom onset, the findings suggest a possible causal relationship. If confirmed, these factors could account for up to 80% of gastric ulcer cases. smoking with a corresponding increase in rate of development of gastric ulcer allowing to become more prominent. In the early 20th century physician's believed they could diagnose ulcers clinically and that the diagnosis required hospitalization for "surgical disease. We show that while smoking and canned materials remained common and virulent in Egypt and the Middle East, environmental changes resulted in changes of the pattern of gastric ulcer producing a change in the manifestations. The consumption of many of canned materials, mainlyFava beans, fava beans, chickpeas and mortadella, increase risk. Many of these canned material contain presentatives cause diseases, which were also inversely related to gastric cancer risk. The intake of smoking was positively associatedwith gastric cancer risk, but primarily in men.

Keywords

Gastric Ulcer, Canned Materials, Smoking

1. Introduction

Gastric ulcers, even in the absence of abnormalities in gastric secretions, have shown a strong association with smoking and canned material consumption. However, while a link has been identified, it remains unclear whether exposure to these factors precedes ulcer development—an essential criterion for establishing causality [1-3]. This study aimed to investigate the relationship between gastric ulcers and these environmental factors prior to the onset of symptoms.

The research focused on chronic gastric ulcers located between the cardioesophageal junction and the pylorus, with symptom onset occurring no more than five years before the interview. The ulcer location was not specified, as the examined factors appeared unrelated to the ulcer's position. To reduce recall bias, the symptom history was capped at five years. Participants with conditions such as duodenal ulcers, hiatus hernia, or esophageal reflux were excluded to ensure symptom relevance to gastric ulcers [4]. The sample was unrestricted apart from these criteria, and 98% of participants

*Corresponding author: abdelrahmanaliter@std.mans.edu.eg (Abdelrahman Alitr)

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had access to a private telephone. Interviews were conducted in 2023 and 2024.

2. Method

Patients:

The study involved 104 gastric ulcer patients (41 men and 63 women) residing in Sydney, diagnosed at the Specialized Medical Hospital. Eligibility required a confirmed diagnosis through endoscopic and histological evaluation, alongside Congalton's status ratings for Sydney suburbs. Fifteen individuals were excluded as controls due to a known or suspected history of peptic ulcers. Community participants were asked about any current or past dyspepsia (persistent or recurring abdominal discomfort) and any prior radiological or endoscopic examinations. Among those approached, 18% reported a history of ulcers or dyspepsia [5, 6].

Exclusion Criteria:

Participants, both patients and controls, were excluded for the following reasons:

1) Limited English proficiency (four patients, five community members).
2) Mental disability (three patients, two community members).
3) Severe physical illnesses (two patients, two community members).

Overall, 95% of the eligible patients and control participants agreed to take part in the study.

3. Data Collection

Participants (patients and controls) were interviewed via telephone, a validated method for data collection. Interviews focused on smoking habits, alcohol consumption, and canned material intake during three periods: (1) lifetime up to the onset of initial ulcer symptoms, (2) five years before symptom onset, and (3) one year before symptom onset. The control group was matched to patients' study periods for consistency [14, 15].

4. Statistical Analysis

This section outlines the statistical methodology and exposure variables analyzed in the study. Below is a summary and interpretation of key points:

4.1. Logistic Regression for Matched Sets

1) Purpose: Logistic regression was used to analyze matched case-control data, emphasizing joint exposure factors while accounting for matching variables like age, sex, and social grade.
2) Assumptions: The model assumes:
   a) The logarithm of the odds ratio is a linear combination of exposure variables.
   b) Effects of exposure factors are cumulative unless significant interactions are found.
3) Model Development:
   a) Terms were added stepwise to the regression equation.
   b) Only terms significantly improving model fit were retained.
   c) Odds ratios and confidence intervals were derived from the regression coefficients and their standard errors.

4.2. Exposure Variables

Four key exposure variables were included:

1) Smoking: Categorized as smoker (≥1 cigarette/day for 6+ consecutive months) vs. non-smoker.
2) Canned Material Ingestion: Heavy consumption (daily for 6+ months) vs. non-eater/light-moderate consumption.
3) Canned Material Usage: Daily user (≥7 times weekly for 2+ months) vs. non-daily user.
4) Non-Eating: Daily user (≥7 servings weekly for 4+ weeks) vs. non-daily user.

4.3. Categorization for Lifetime Period

Canned material users during their lifetime were further classified into:

1) Non-daily users: Less than one serving daily or one to five servings daily for <2 months.
2) Light/moderate daily users: One to five servings daily for ≥2 months or >5 servings daily for <2 months.
3) Heavy daily users: >5 servings daily for ≥2 months.

4.4. Regression Models

1) Lifetime and Five-Year Prior Periods:

Simple main effects models were used.

Odds ratio: log (odds ratio)=cx(smoking)+b(canned materials)\text{log (odds ratio)} = c_x (\text{smoking}) + b (\text{canned materials})log (odds ratio)=cx(smoking)+b(canned materials).

2) One-Year Prior Period:

The model included an interaction term between smoking and canned material ingestion, reflecting combined effects.

4.5. Exposure Quantification

1) Exposure levels were quantified using metrics such as:

Pack year: Smoking 20 cigarettes/day for one year.

Nip year: Consuming one canned material daily for one year.

Tablet year: Eating one canned serving daily for one year.

2) Fractional values (e.g., 0.5 pack years for 10 cigarettes/day for one year) allowed more precise quantifi-

cation.

4.6. Population Attributable Risk (PAR)

1) Definition: PAR represents the proportion of the disease attributable to the studied exposures and quantifies the potential reduction in disease prevalence if these exposures were eliminated [7, 8].
2) Method: Whittemore's method was used to calculate PAR as a percentage based on exposure frequencies among patients and controls.

Significance of Methodology

The approach ensures:

1) Accurate modeling of exposure effects within matched sets.
2) Distinction between the independent effects of variables and their interactions.
3) Robust estimates of disease risk and the public health impact of exposures.

This statistical framework provides a strong basis for analyzing risk factors like smoking and canned material consumption while accounting for complexities in the data [16-19].

5. Results

Independent analyses showed significant associations between smoking, daily canned material consumption, and gastric ulcer risk across all periods ($p < 0.01$). However, canned material usage alone was not a significant risk factor ($p > 0.05$). Key findings included:

1) Smoking and daily canned material consumption were significant risk factors, whether occurring alone or together.
2) An interaction between smoking and canned material consumption one year before symptom onset showed a negative effect, reducing ulcer risk when combined ($p < 0.05$).
3) No interactions were found between matching variables (e.g., age, sex) and exposure variables [20, 21].
4) A dose-response relationship was observed for canned materials ingestion, with higher consumption linked to increased ulcer risk.

These results suggest significant roles for smoking and canned material consumption in gastric ulcer development, with interactions modifying their effects in certain periods [11, 12].

6. Discussion

This study identified a strong link between gastric ulcers and smoking and canned material consumption prior to symptom onset. Several criteria for causality were evaluated:

1) Temporal Relationship: Exposure to smoking and canned materials preceded symptom onset, fulfilling a key condition for causation [13].
2) Strength and Consistency: Associations were strong across study periods, with minimal recall bias due to the limited symptom history. Past research supports these findings.
3) Biological Gradient: A dose-response effect was evident, particularly for canned materials, aligning with known health impacts.
4) Biological Plausibility: Smoking and canned materials are linked to gastric mucosal damage and increased acid secretion, supporting their role as ulcer risk factors.

Adherence to these causality criteria supports the conclusion that smoking and canned materials are significant risk factors for gastric ulcers. Eliminating these exposures could significantly reduce disease prevalence, akin to the impact of smoking cessation on lung cancer rates [9, 10].

Abbreviations

PAR	Population Attributable Risk
OR	Odd Ratio
RM	Regression Model

Conflicts of Interest

The authors declare no conflicts of interest.

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